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Synopses

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it's time for change

a discussion of the diagnosis and classification of caries in children and adolescents

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Introduction

The youth of today have lower caries prevalence and incidence rates than every generation before them.[1,2] The face of decay has changed.

New diagnostic criteria will be required to identify the more subtle forms of disease. New classification systems keyed to treatment modalities are being devised.

This essay will discuss traditional methods of diagnosis in younger patients. It will identify the problems that the new pattern of juvenile decay has created and new diagnostic strategies that have been formulated to overcome these. It will address old and new systems of caries classification.

Diagnosis in Children and Adolescents

Traditional caries diagnosis is visual-tactile, utilising clinical examination and routine radiography. In Europe, examination involves visual inspection of the teeth alone.[3] In contrast, Northern America still uses the sharp explorer advocated by GV Black for detecting sticky pits.[4] The Pacific basin continues to utilise both approaches. Visual inspection alone takes over twice as long as tactile examination.[4] It is twice as efficient at detecting caries.[5] Because lesions are progressing more slowly, more time is available for demineralised enamel to be damaged by the explorer and cavitation therefore promoted.[1] The explorer has been said to transplant cariogenic bacteria from caries-active to caries-inactive sites, but this has yet to be proven.[3] The current world trend is toward entirely visual inspection of clean, dry and well illuminated tooth surfaces.[1] This examination looks for changes in enamel translucency, dentinal shadowing or frank cavitation.[1] Diagnosis will often require multiple examinations over time and the utilisation of several methods of visual inspection in order to reduce the number of false positive diagnoses as far as possible. Visual inspection is important because the majority of juvenile decay is located in the pits and fissures and is therefore able to be visually diagnosed.[6]

Clinical examination is routinely augmented by radiography. Posterior bitewings can be particularly difficult to obtain for younger patients and so anterior occlusal, lateral cephalometric

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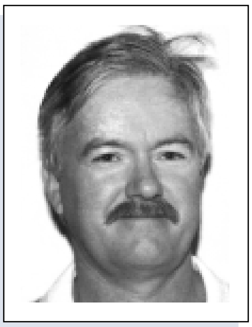

president's report

Dear Members,

The commencement of a new position, particularly a professional one in today's free market economy, brings with it both challenges and expectations that appear somewhat daunting.

There is increasing pressure placed upon us to respond to criticism and accusation levelled on our profession by a well informed and Internet literate public. Only the other day, a mother demanded I make dentures for her two-year-old son with Ectodermal Dysplasia and multiple missing teeth. Apparently, someone on a net chat board had said that that age 2 was the right time to start. The fact that I could not even examine the child without parental restraint seemed to make no difference to the mother's request.

Dentistry has been accused of both protectionism and self-serving behaviour with regard to both professional practice and conduct. The electronic and print media have recently latched onto dentistry, particularly dentistry for children, as the latest fashionable health establishment to attack. A sceptical public is watching our reaction very closely.

Care about our patients -

children can sense compassion in how we deal with them.

Heal the wound -

try to meet the immediate concerns of the child rather than what we think they need.

Invite participation -

involve the child and the parent in the planning and caring process.

Listen -

good listening skills is the key to good communication.

Dialogue -

develop a thoughtful and personal dialogue with the child.

Render only care that is needed -

good health care is determined by the least number of procedures.

Encourage expression of feelings -

if there is a problem, find out what it is.

Nuture the relationship -

children and parents want to see you and no amount of electronic gadgetry can replace a caring dentist.

Unfortunately, our knee jerk response to public criticism is often official statements that lack personal depth or deny the problem altogether. This immediate "damage control" can often damage any favour we might have with an already cynical press and public.

Sensationalism aside, we need to become more self critical and ask ourselves whether there is any underlying truth to the claims which have been levelled against us. We might also need to redefine our own professional behavioural guidelines. Dentistry's positive public image and high esteem have been built over many decades on a professional dedication to pain relief and ongoing sympathetic health care.

The underlying message from most of the sensational press headlines that I read is not to lose touch with the children we care for and don't forget that we have been entrusted with their oral health. Those practitioners that are perceived to care for their patients are less likely to be sued even when things go wrong. Take care of our patients or someone else will take care of us!

The challenge for all dentists with an interest in children is to retain these basic caring and communication skills in a world of technological change and advancement.

Lets put our patients first and work together for the common good of children and the profession. With all your help, the task at hand seems a little less daunting.

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and panoramic views are often substituted. The dose of radiation must be kept as low as is reasonably possible: the so-called ALARA principle. It has been questioned whether it is still justifiable to continue the practice of routine radiographic screening following the decline in caries prevalence.[3] However, bitewing radiographs have been shown to reveal dentinal lesions in up to 30% of adolescent permanent molars which had been clinically considered to be sound.[7] The normal dose of radiation per bitewing is about 2.2mGy and therefore minimal.[8] Roeters *et al* has investigated risk factors that may be used to time radiography for children and therefore limit their exposure.[9]

Nonetheless, a somatic stochastic effect of radiation is inherent to the procedure. There is a high incidence of overlapped and therefore unreadable surfaces.[10]

Murray and Shaw have shown that 21% of all lesions are not apparent on radiographs.[10] These points combined with the 'hidden caries' phenomenon of the 1990s, whereby the majority of lesions develop under a visually intact enamel surface, are causing a re-appraisal of the need to obtain radiographs of our paediatric patients. The benefits of detecting the slowly progressing, clinically undetectable lesion currently outweigh the costs of radiography.

Diagnostic Dilemmas

The change in caries rate and pattern is challenging our current approach to caries diagnosis in children and adolescents. Traditional techniques produce a constant rate of false positive diagnoses.[1] Because the majority of teeth examined today are caries-free, this reduced prevalence has resulted in an increase in the number of false positive diagnoses made.[1] New diagnostic techniques with increased sensitivity are needed to overcome this problem. It has been reported that the worldwide decline in both caries prevalence and incidence is ending and caries is again changing it's pattern.[11]

The youth of today have lower caries prevalence and incidence rates than every generation before them

The change in lesion morphology is testing these traditional diagnostic methods. Occlusal lesions in particular are more apt to present with intact enamel surfaces than ever before.[1] Often the hardest to diagnose, incipient lesions are accounting for a greater proportion of total diagnoses made. This difficulty is confounded by the high incidence of fluorosis in our societies which can appear visually similar.

Finally, the traditional methods of caries diagnosis have not been technologically assessed. New systems are being evaluated, but it must be appreciated that the older systems have not been judged on their ability to diagnose caries.[3] The need for improved diagnostic methods is underscored by the low level of agreement between examiners utilising the traditional methods of diagnosis.[12]

New Approaches to Caries Diagnosis in Children and Adolescents

The predictive value of our traditional diagnostic systems has diminished and so new tests are being developed and evaluated. Fiberoptic transillumination (FOTI) has been used by some countries for years but has had little use in others.[3] It involves illumination of the posterior approximal contacts by a fiberoptic light source.[3] It has no biological effects on the patient, unlike radiography.[3] Recent studies indicate that the sensitivity of FOTI is comparable to that of radiography.[13,14,15] Verdonschot *et al* has concluded, using drilling as validation, that FOTI is superior to

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clinical examination and radiography for detecting small occlusal lesions.[16] A histologically validated study has shown that FOTI is superior to clinical examination and radiography for both shallow and deep occlusal lesions, while radiography alone is better at diagnosing deep lesions.[17] Conversely, several authors have stated that radiography is diagnostically superior to FOTI.[18,19] Stephen *et al* conducted a prospective, large scale clinical trial over three years and concluded that radiography was better at diagnosing caries than FOTI.[18] This study stated that FOTI should be used as an adjunct to, rather than replacement for, clinical radiographic diagnosis. Interexaminer variability has been shown to be higher for FOTI than for radiography.[20] The conflicting nature of this evidence indicates that further research is required before this technique becomes routinely used in practice. In particular, the prognostic value of FOTI for approximal lesions must be evaluated.

Electrical resistance measurement (ERM) is another older technique that has been revived. ERM assesses the integrity of an occlusal surface by it's electrical conductivity.[21] Intact surfaces have little or no conductivity, while the conductivity of demineralised surfaces increases as they lose mineral.[22] The effect of the resistance probe on the tooth surface has not been investigated, but the method itself is believed to have no adverse effects on the patient or the dentist.[21] Several studies show that ERM has the same diagnostic accuracy as radiography and clinical examination for diagnosing occlusal caries.[22,23,24] In a drilling-validated study, ERM was shown to be more sensitive than clinical examination and to have higher specificity than radiography.[25] While early investigation suggests ERM is at least as good as radiography for diagnosing caries, further investigation is warranted if it is to be routinely employed. The elective and temporary separation of approximal tooth surfaces dates back to last century.[3] Separation is achieved by orthodontic elastomerics rather than the painful immediate separation that was previously used.[26] It is currently being taught in many British and European dental schools as a diagnostic adjunct in equivocal situations.[3] It has been shown to be both practical and viable in children.[27] It is conservative. However, the logistical problem of a second visit 3-7 days after the placement of these elastomerics is a disadvantage.[3] Broken components will delay diagnosis. In addition, approximal cavitation is occurring later in the disease process and, coupled with dental fluorosis which can look the same, simple white spot demineralisation may be difficult to detect in such visually limiting spaces.

the worldwide decline in both caries prevalence and incidence is ending and caries is again changing it's pattern

Digital radiography is a new approach to caries diagnosis. The computerised image is obtained by digitisation of a conventional radiograph or direct digital transfer.[28] The latter approach has the advantage of a reduced dose of radiation due to increased sensitivity of the receptor compared with the film.[29] Both methods can be visually enhanced so that where a conventional film may be underexposed and thus diagnostically useless, a digital image may be enhanced and become serviceable.[29] Contrast enhanced digital radiographs possess higher diagnostic sensitivity without a increase in false positive diagnoses relative to conventional radiographs of non-cavitated teeth.[30,31]

Digital radiographic filters are being used to delineate between diseased and sound tissue and therefore estimate the depth of a lesion.[32] These filters tend to result in lost detail.[21] The diagnostic accuracy of such filters has yet to be investigated.[21]

'Digital subtraction radiography' has been developed to aid in the diagnosis of non-cavitated surfaces.[28] This technique assumes that 'atomic

noise' can be eliminated through superimposition of identical structures that are compared to each other.[21] Wenzel and Halse have used this method to show an increase in the density of occlusal lesions after the application of topical fluoride.[33] This is a reliable sign that a dentinal lesion is present.[21] No false positive diagnoses were observed for this technique, but 13 were made for conventional films.[33] In summary, digital radiographic techniques use a low dose of radiation and are proving to be as accurate as conventional film. They have sufficient resolution for the diagnosis of caries. Image enhancement may increase the accuracy of the film, although paper prints are less accurate than monitor images.[21]

Simonsen states that binocular magnification makes all the difference when diagnosing caries.[34] Ismail *et al* has suggested that magnification may aid in the diagnosis of very early caries and therefore limit the intervention that is required.[35] This hypothesis has yet to be tested. This advantage may be offset by the tendency to overtreat.[35] Whitehead and Wilson report 56% more decisions to intervene when using this technology.[36] They conclude that clinicians should review the criteria on which they base the decision to intervene before they use magnification.

Lactobacilli and *Streptococcus mutans* counts are being used to diagnose caries. Because these bacteria are implicated in the aetiology of caries, tests which measure the pH drop associated with the metabolism of these micro-organisms are being used to predict and diagnose caries activity.[37] Caries activity tests are available, but rarely used in practice.[4] Nishimura *et al* has shown that these tests positively correlate with the level of *Lactobacilli* and *S. mutans* in the mouth.[38] Nishimura has also shown that these tests are highly sensitive screening methods for primary approximal caries.[38] However, while the level of *S. mutans* may indicate caries risk, the number of *Lactobacilli* may only be a good index of dentinal caries and not caries risk or enamel caries activity.[38]

Youth decay has traditionally been classified in the same manner as adult decay

The extent of dentinal caries may be diagnosed with a disclosing dye. Dyes, such as acid red or 0.5% fuchsin in propylene glycol, stain denatured collagen and bacterial polysaccharide.[39] Because the inner three zones of a carious lesion are believed to be sterile, the aim of these dyes is to distinguish affected dentine from infected dentine.[40] Enamel dyes have not yet been developed.[4] These would be especially advantageous in detecting the early lesion and therefore enabling minimal intervention or fortuitous preventative therapy to be instituted. It is worth noting that some dentists have stated that routine use of these dyes would cause excessive removal of tooth structure and increase the likelihood of pulpal exposures.[40]

The activity of the lesion must also be diagnosed. Nyrad and Ferjerskov have concluded that microbiological methods for the evaluation of caries activity are currently unreliable.[41] Hojo *et al* states that the dentinal acid profile bears a clear relationship to the activity of the lesion.[42] Both these techniques require further research.

Current Classification of Caries in Children and Adolescents

Youth decay has traditionally been classified in the same manner as adult decay. Many systems have been devised and, as our knowledge of the carious process has grown, just as many systems have become obsolete. One of these early systems classified decay according to the diseased tooth surface. Caries was either smooth surface, occlusal, approximal or, very rarely, secondary. This system was simple but failed to address lesion depth, activity, size, which tissues were infected or treatment strategy.

Likewise, GV Black classified decay according to the site that had been undermined and the cavity design required to restore it.[43] This system was simple but failed to cover the points previously made. This system is redundant because it fails to classify the remineralisable, incipient lesion that is currently making up a large proportion of total diagnoses made. Instead it presupposes the need to restore every lesion.

Caries has been described by the number of teeth involved. The term 'rampant decay' has been replaced with 'adolescent' or 'early childhood decay'. These names describe the typically large numbers of teeth infected over a short period of time. This system clearly identifies the severity of the

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problem and the complexity of the response required. It does not provide any information on the number of diseased teeth, which teeth are involved or the characteristics of the lesions. This system should only be used when augmented by a more detailed description of the problem.

Caries may be classified according to the tissue undermined by the disease. Under this system caries is enamel, dentinal or pulpally involved. Such a system indicates the degree of disease progression and therefore gives some idea of the way that the disease may have spread from its point of origin. The system does not address the size of the lesion or the integrity of the tooth surface and therefore gives no indication of the appropriate treatment. Radiographs will usually be required to classify these lesions and, because these tend to underestimate the progression of disease, the basis of this system may be undermined. Nonetheless, many paediatric clinics still use this system today.

A more detailed version of this system exists. Classified according to radiographs, this scale labels minimal enamel lesions as R1, full thickness enamel lesions as R2, minimal dentinal lesions as R3 and full thickness dentinal lesions as R4. This system has the same advantages and disadvantages as that chronicled above. It is widely used both in paediatric and adult clinics. Caries may be classified according to the activity of the lesion. Arrested lesions have rarely been seen in young people but their numbers are beginning to increase in today's children and adolescents.[1] This may be a result of an increase in water fluoridation, use of discretionary fluorides, and dental awareness. This system should be used as a adjunct to other classifications because it is inadequate to fully the describe the lesion.

The diagnosis and classification of caries in children and adolescents is undergoing a revolution

Caries may be graded as incipient, early, cavitated or pulpally involved. This is probably the most helpful paediatric classification in use today. This system provides us with information regarding the two most important things to know about a lesion: whether the enamel surface is intact and the degree of progression. The site of the lesion is not implied, but this is a secondary consideration to the above points.

New Systems to Classify Caries in Children and Adolescents

We require new classification systems because dental caries has changed the way in which it presents. Pitts and Longbottom have recently devised a system based on the appropriate management of the lesion.[44] It contains only two categories: OC (lesions for which **O**perative **C**are is advised) and NO (lesions for which **N**on-**O**perative care is recommended). Many European countries have already adopted this approach, albeit with different terminology.[3] This system is simple and acknowledges the relationship between diagnosis and treatment planning. It considers the size, severity, prognosis, site of attack, activity, status of the enamel surface and which tissues are diseased as presuppositions to classification. It forces the clinician to think about each lesion as a whole rather than plan their treatment on the basis of some predetermined classification code/implied treatment structure. However, the complex consideration behind this system may prevent it from being widely used. Additional information should be recorded of each lesion for medico-legal reasons and for future reference. This task may be time consuming and laborious.

Similarly, Nyrad and Ferjerskov have classified caries according to the activity

of the lesion.[45] Arrested caries is coded as NOTAL (**NO** **T**reatment **A**t al**L** required) and active lesions are classified as either NOT (**N**on-**O**perative **T**reatment) or OT (**O**perative **T**reatment). The NOT category is similar to Pitts and Longbottom's NO (**N**on-**O**perative care advised) code, however the former acronym acknowledges that non-operative intervention is a type of treatment itself.[44] The advantage of this system is that it is simple: caries activity may be directly linked with the appropriate treatment.

Mount and Hume have published a system that recognises how a lesion may be restored with modern materials and techniques.[46] They recognise that the GV Black classification is outdated because it was devised at a time when caries was rampant and the carious process was poorly understood. Their system codes 12 types of carious lesion by two digits each. The first digit of every code indicates whether the decay is located in the pits and fissures, approximal contact points or about the cervical of the tooth. The second digit recognises the size of the lesion. This may be just beyond remineralisation (minimal), a larger lesion (moderate), a lesion that has undermined the cusps of the tooth (enlarged) or where the cusp has already been lost (extensive). This system acknowledges the increasing complexity of the enlarging lesion. It is readily computerised. Unlike the GV Black classification, it addresses the size of the lesion. It identifies the diseased site. It does not account for the activity of the lesion and therefore presupposes the need to restore. Under this classification there is no category for incipient lesions that require preventative therapy alone.

Conclusion

The diagnosis and classification of caries in children and adolescents is undergoing a revolution. Clinicians are finding that old diagnostic and classification systems have limited utility in considering the modern forms of paediatric caries. New methods are being developed to counteract this and preliminary research has indicated that they have great potential. It is an exciting time to be caring for young people. Today's dentists must not only fuel the drive for new and better diagnostic and classification systems, but grasp the responsibility of providing these children with teeth for life.

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inter-relationship between the upper airway and maxillofacial growth

multi-disciplinary treatment approaches

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Introduction

Upper airway obstruction during childhood that is sufficient to cause mouth breathing may be associated with altered maxillofacial development.[1,2] For this reason, mouth breathing has been used to justify various dental and surgical procedures that aim to reduce upper airway obstruction, promote nasal respiration and more normal dentofacial development. However, a clear cause and effect relationship between mouth breathing and maxillofacial development has not been established. This essay will examine the inter-relationship between the upper airway and maxillofacial growth and comment on the multidisciplinary treatment approaches involved.

Relationship between the upper airway and maxillofacial growth

To understand the upper airway affect on maxillofacial growth, it is important to review the growth patterns involved in normal development. Growth of the nasomaxillary complex is produced by two basic mechanisms; active growth of the maxillary sutures in response to the surrounding tissues, and passive displacement of the maxilla created by growth of the cranial base.[3] As the maxilla is translated downward and forward, bone is deposited at the sutures and at the same time, surface remodeling removes bone from the anterior surfaces.[3]

The longitudinal implant studies of Bjork et. al., in the 1960's demonstrated the extent to which the maxilla and mandible rotated during growth.[4] The pattern of vertical face development is strongly related to the rotation of the jaws. For an average individual with normal vertical facial proportions, there is about minus 15 degrees of internal rotation (rotation of the core of the mandible) from age four to adult life. This internal rotation is not apparent in the average individual, as external rotation (surface changes) tend to camouflage the change. Maxillofacial growth patterns however, are sensitive to environmental influences such as the upper airway and mode of respiration.[4]

Various animal and human studies have been conducted to determine the association between the upper airway and maxillofacial growth.[1,5-7] Longitudinal studies in primates by Harvold and Miller have assessed the effects of experimentally induced mouthbreathing,[5,8,9] revealing mouth breathing can influence lip, tongue and mandibular position. It was proposed that this influenced craniofacial growth. The craniofacial complex of these primates showed anterior divergency associated with open bite and variable molar relationships.[5]

Results from these primate studies must be read with caution as total nasal obstruction was produced. The primate was not structured to mouth breathe and an exaggerated postural response to open the oral airway was necessary for survival. It should also be noted that both neuromuscular and skeletal changes in this study showed a wide range of response. [5,8,9]

For humans, certain conditions such as choanal atresia may exist where total nasal obstruction occurs, however human studies have indicated that total nasal obstruction is rare.[10-13] The most common respiratory mode is a simultaneous oral and nasal airflow.[10] Animal studies should therefore focus on whether partial nasal obstruction can alter maxillofacial growth.

This was recently addressed by Yamada et. al.,[14] who examined the effect of artificial nasal obstruction on craniofacial growth in young *Macaca fuscata* monkeys.[14] Nasal respiratory obstruction was created by injecting dental impression material into the posterior wall of the nasopharynx under general anaesthesia. Craniofacial growth in the experimental monkeys was compared to a control group by means of cephalometric analysis. The results illustrated nasopharyngeal obstruction was associated with downward and backward rotation of the mandible, with upward and backward growth of the condyle resulting in anterior open bite.[14]

The clinical studies of Linder-Aronson stimulated interest in a possible relationship between nasorespiratory function and

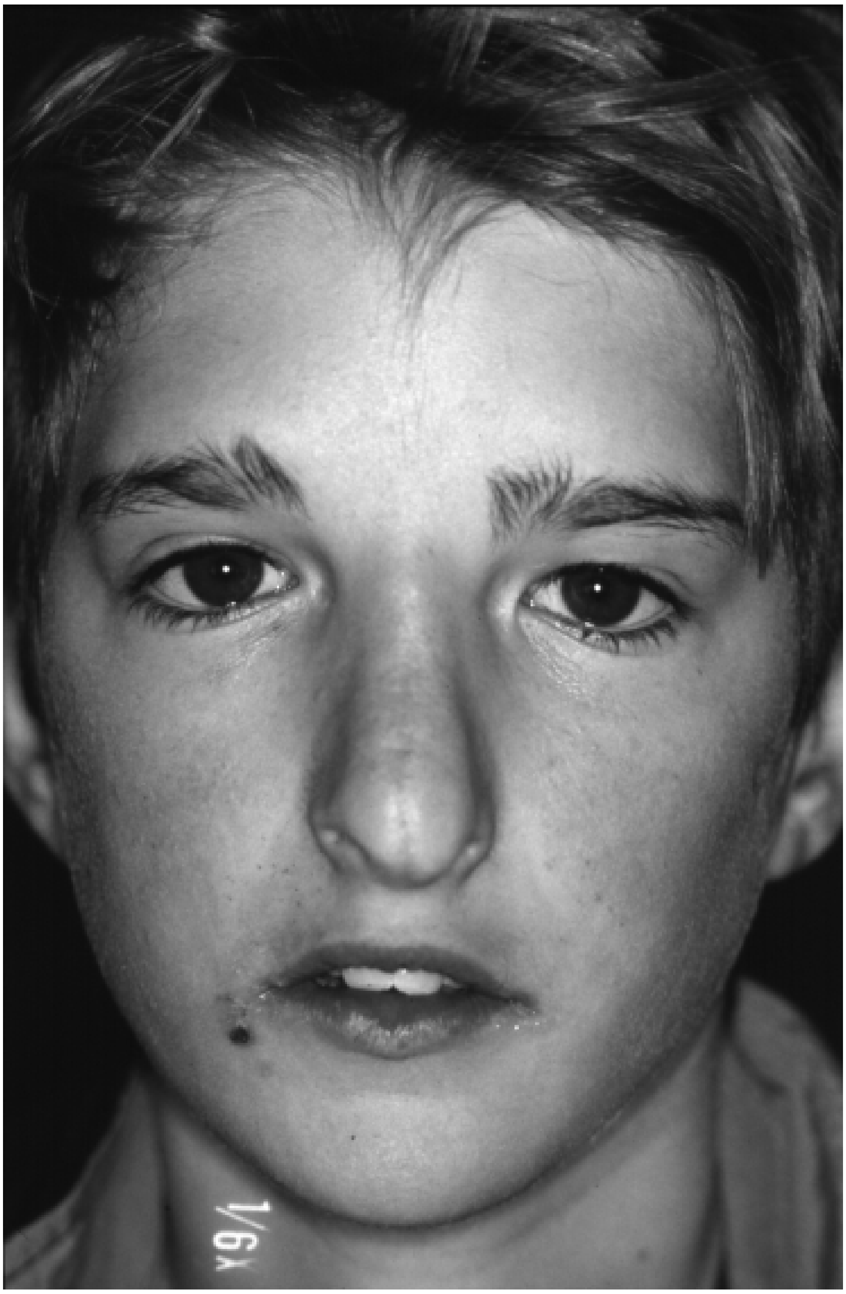
craniofacial growth.[1,6] Patients classified clinically as mouthbreathers were seen to have several significant deviations in dentofacial morphology.

Linder-Aronson postulated that adenoidal tissue influenced the mode of respiration and as a result altered craniofacial growth in human subjects.[1] Three changes in posture are believed to cause this malocclusion: lowering of the mandible, positioning the tongue downward and forward and extending the neck and head.[1,6]

The typical features considered to be associated with mouth breathing are commonly referred to as 'long face syndrome' or 'adenoid facies'. An example is given in Figures 1-4.

In this condition there is a downward and backward rotation of the mandible during growth. This is accompanied by excessive eruption of posterior teeth, increased lower face height, lip incompetence, narrow alar base, narrow maxillary arch with a high palatal vault, retroclined incisors, a posterior cross-bite and a Class II dental malocclusion.[1,15,16]

Figure 1



In a later study, Linder-Aronson, Woodside and Lundstrom, investigated craniofacial growth after adenoidectomy in human subjects with mouthbreathing.[6] Changes in breathing mode, mandibular growth and proclination of incisors were measured five years post adenoidectomy. Although individual variations were evident, the study demonstrated that adenoidectomy generally corrected the abnormal pattern of mandibular growth produced by adenoid hypertrophy.[6]

Despite these findings, the effect of breathing pattern on maxillofacial growth, is still subject to debate. Some controversy stems from our inability to define mouth breathing. Clinicians sometimes classify patients as 'mouth breathers' or 'nasal breathers', inferring that people either breathe entirely through their mouths or entirely through their noses. This oversimplification neglects the spectrum between these two points where the majority of people fall. Controversy also remains about the validity of current methods used in quantifying the oronasal breathing ratio, as well as the effect relationship between oronasal breathing and postural changes resulting in altered maxillofacial growth.[17,18,19-21] In addition, only three studies in the medico-dental literature have experimentally studied postural changes after nasal obstruction in humans.[22-24] Studies on respiratory mode in children have so far been cross-sectional and therefore not addressed

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the relationship of maxillofacial growth changes and respiratory mode.[25]

Methods used to differentiate between nasal and oral breathing have proven to be poor predictors of respiratory mode.[13,26,27] Historically, respiratory mode was determined by clinical impression with minimal success.[28] Patients were classified as mouth breathers on the basis of morphologic criteria such as 'adenoid faces', 'incompetent lips', questionnaires, condensation on cold mirrors and visual inspection of the upper airway. Clinical criteria of mouth breathing does not accurately identify children who breathe through their mouth, some patients show an open mouth tendency even though the nasal adequacy is normal.[29] Cephalometric radiographs and rhinomanometry to evaluate nasal obstruction have been available for several decades. Although a positive correlation between airflow and airway measurements has been demonstrated using cephalometric radiographs, the three-dimensional aspects have been neglected as two-dimensional images are unable to illustrate transverse dimensions of the nasopharynx.[30]

Posterior rhinomanometry has been employed to measure both nasal power and nasal airway resistance.[13,30] The rhinomanometer consists of an airtight facemask that covers the nose, with two catheters measuring the pressure drop between the nose and the mouth. One is placed in the posterior oropharynx with a tight lip seal, the second within the facemask. Results of these studies revealed that oral and nasal components of respiration could not be associated with either power or nasal airway resistance.[13,30] The results suggest that the probability of such tests identifying increased nasal resistance is not significantly better than random selection.[13,26,27] More importantly, these studies also found that nasal resistance is not a reliable predictor of reduced nasal respiration.[28]

Evaluations of breathing modes of growing children on different days shows variations in respiratory activity.[30] Nasal breathing cycles of one to several hours have been found to occur in 80 per cent of adults and the mode of breathing may change according to the time at the collection of data.[31,32]

Awareness that the airway is being assessed is an inherent difficulty in these evaluations. The presence of a mouthpiece in place during examination could easily influence a person to increase the oral component of breathing. This has since been confirmed in a study of an adult population by Hairfield et. al.[33]

Treatment approaches of the upper airway

The most important aspect of treatment modalities is that of efficacy.[28] Do the benefits of such treatment outweigh the costs or risks? If so, is the change in incisor position or mandibular growth going to make the malocclusion easier to treat? If the occlusal benefits are unlikely, the value of the procedure in orthodontic terms should be reconsidered.[28]

An important issue in nasal obstruction is the ability to identify where the obstruction occurs. The nose has an anterior opening (the nares), a middle portion that is influenced by the turbinates and their associated vascular changes which are influenced by infection and allergy and the posterior nares that open into the nasopharynx and may be affected by adenoidal hypertrophy. Nasal allergy is the most frequent cause of upper airway obstruction, indicating a more conservative approach. Management consists of a wide spectrum from normal saline nose drops or topical steroid nasal sprays for perennial rhinitis through orthodontic appliances to complex surgical procedures.

Rapid Maxillary Expansion

Rapid maxillary expansion (RME) has been recommended for the correction of transverse maxillary deficiencies with the additional benefit of increasing nasal airflow. The technique of RME was originated by Angel in 1860. In 1886 Eysell, an otorhinolaryngologist, proposed that the lateral expansion may relieve nasal obstruction. Since this time, there has been a long-standing controversy over the efficacy of RME in improving nasal respiration.

During the course of RME, the maxillary and palatine bones are disarticulated

along the mid-palatal suture and move laterally. These movements are rotatory the axis being beneath the cranial base with the most anterior and inferior parts moving furthest.[34] The expansion carries the lateral walls of the nasal air passages outwards increasing the trans-alar width.

The nasal valve in the normal nose presents the smallest nasal cross-sectional area and provides the most significant airflow resistance during breathing. The valve is in the region between the upper and lower lateral cartilages and the pyriform aperture, just beyond the anterior ends of the inferior turbinates. Aerodynamic studies and cadaver dissections have found the cross-sectional area of the nasal valve to be between 0.3 and 0.4 cm^2 in each nostril.[35,36]

Warren studied the effects of RME and surgical expansion on nasal cross-sectional area.[36] Patients treated with RME resulted in an increase in nasal cross-sectional area of 45 per cent.[36] Similarly, those patients treated with a total maxillary segmental osteotomy increased the cross-sectional area by approximately 55 per cent post-operatively. Despite a general increase in airway patency approximately one third of the subjects in both groups did not achieve enough improvement to eliminate the probability of obligate mouth breathing.[36]

Nasal airway resistance (NAR) using posterior rhinomanometry in 26 patients receiving RME was studied by Timms.[34] Reductions in nasal airway resistance were recorded in all cases with an average of 36.2 per cent. The correlation between the resistance reductions and the expansion was however weak. One cannot therefore predict with any accuracy how much expansion would be required to reduced NAR by a given amount. The greatest reduction in NAR occurred in patients with high pre-treatment NAR values which was supported in an earlier study by Hershey et. al.[37] It is probable that Poiselle's Law applies to this situation. "The airflow varies with the fourth power of the radius, in other words, doubling the width of the nasal cavity increases the flow 16 times. Although Timms has shown a reduction in NAR, it is clearly not a predictable decrease. Hartgerink, Vig and Abbot, have also shown that despite a reduction in NAR following RME, this did not change the respiratory mode of the patient.[38] The difficulty in treatment therefore lies with the inadequacy of determining which patients will respond to therapy.

Obstructive sleep apnoea syndrome (OSAS)

Cessations of breathing for ten seconds or longer are termed apnoeas. When thirty or more apnoeic episodes occur in the course of seven hours sleep which results in excessive sleepiness during the

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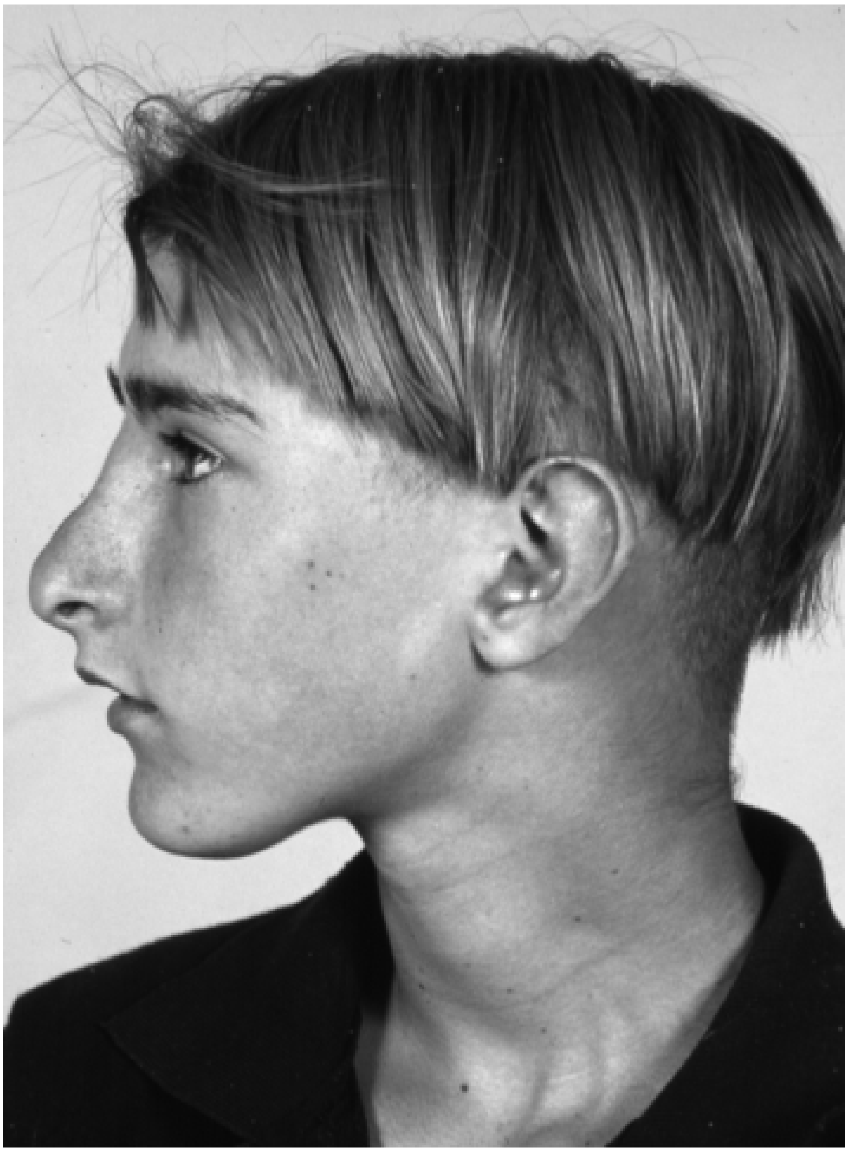
Figure 2



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waking hours, a person is described as having obstructive sleep apnoea syndrome (OSAS).

OSAS has recently assumed greater importance in the dental literature. Aetiologic or predisposing factors include gender, body mass index (BMI), anatomically narrow airway, craniofacial deformities, muscular hypotonia, the Bernoulli effect, sleep posture and fatty deposits in the tissues of the upper airway. A variety of treatments ranging from conservative to surgical have been suggested, those of particular interest to the dental profession include mandibular protrusion appliances and orthognathic surgery.[39] Surgery was the primary form of therapy for OSAS until Sullivan et al, reported the first successful treatment of OSAS with nasal continuous airway pressure (CPAP) in 1981.[40]

Tracheostomy

Permanent tracheostomy was the first surgical procedure performed on patients with OSAS. Tracheostomy has a success rate of almost 100 per cent because it bypasses all of the potential obstructive sites in the upper airway. Despite its effectiveness, almost all patients experience psychological depression from the associated social and medical problems. The procedure also leaves patients aesthetically disfigured and places them at risk of local complications such as bleeding, infection, pain and granulation tissue formation.[39] Tracheostomy may however be used as an interim treatment until other definitive surgical procedures are completed.

Tracheostomy is also used in the emergency treatment of neonates with Pierre Robin sequence (PRS). The clinical triad of PRS comprises micrognathia, glossoptosis and respiratory obstruction associated with an incomplete cleft palate illustrated in Figs 5-6.[41] The palatal cleft in PRS is believed to result from mechanical obstruction of the tongue secondary to either mandibular hypoplasia or compression of a normal-sized mandible against the developing thorax.[41]

There is a wide variation in the severity of PRS, which presents in the neonatal period with upper airway obstruction and feeding difficulties. These symptoms gradually subside with mandibular growth and improved control of the tongue. However, in neonates with severe micrognathia, failure to thrive due to chronic airway obstruction or severe respiratory distress, the situation becomes potentially fatal. A tracheostomy to relieve the obstruction in these cases is often indicated.[42]

Figure 3

Uvulopalatopharyngoplasty (UPP)

Uvulopalatopharyngoplasty (UPP) was first described in 1964 for the treatment of habitual snoring by Ikematsu. The procedure designed to increase the potential airspace in the oropharynx, involves tonsillectomy, adenoidectomy, excision of the uvula and redundant lateral pharyngeal wall mucosa, with resection of eight to 15 mm along the posterior border of the soft palate. Significant complications of UPP include nasal regurgitation, hypernasal speech and palatal stenosis.

Other surgical procedures

Other surgical procedures include partial glossectomy in cases of macroglossia, and nasal surgery. Nasal obstruction may be caused by a deviated nasal septum, nasal polyps or enlarged turbinates. This is managed by septoplasty, nasal polypectomies or inferior turbinectomies and are helpful adjunctive surgical procedures to decrease nasal airway resistance in the treatment of OSAS.

Non-surgical management

The most commonly used treatments in the non-surgical management of OSAS include weight reduction, nasal CPAP, and dental appliances. All of these modalities require high levels of patient compliance for successful outcomes.[39] Nasal CPAP is the most effective form of non-surgical therapies for OSAS, however poor long-term patient compliance results in a variable success rate from 54 to 75 per cent.[39] Nasal CPAP is cumbersome, cosmetically unappealing and noisy. Other causes of non-compliance with nasal CPAP include mask discomfort, rhinitis, eye irritation, dryness of the nose and throat, skin rashes and chest discomfort.[39] Long-term compliance rates with dental appliances are lower than those reported for nasal CPAP, ranging from 48 to 52 per cent. Appliances are designed to bring the mandible and tongue forward, opening up the lower pharynx to allow unrestricted breathing.[39]

Orthognathic surgery

Mandibular osteotomy

Since a mandibular osteotomy performed to correct a Class III malocclusion carries the tongue posteriorly, it is feasible that the upper airway could be compromised at the time of surgery and in the immediate post-operative period. Clinically, it is well known that respiratory compromise is rarely experienced in association with a mandibular set-back procedure. Several studies have presented data that indicate the extent of the reflex physiologic adaptations which occur to ensure the airway is maintained.[43,44] The hyoid bone and posterior tongue move inferiorly and posteriorly initially following mandibular set-back and then slowly move anteriorly toward their pre-operative positions.[43,44]

Maxillomandibular advancement has proved successful in the treatment of OSAS, however, the exact anatomical change responsible for curing OSAS is unknown.[44] Since the upper airway and pharynx are dynamic structures and extremely variable specific measurements with radiographs are difficult to achieve in evaluating preoperative and postoperative changes.

Le Fort I Maxillary Impaction

When superiorly repositioned, the maxilla encroaches on the nasal cavity, therefore reducing its volume.

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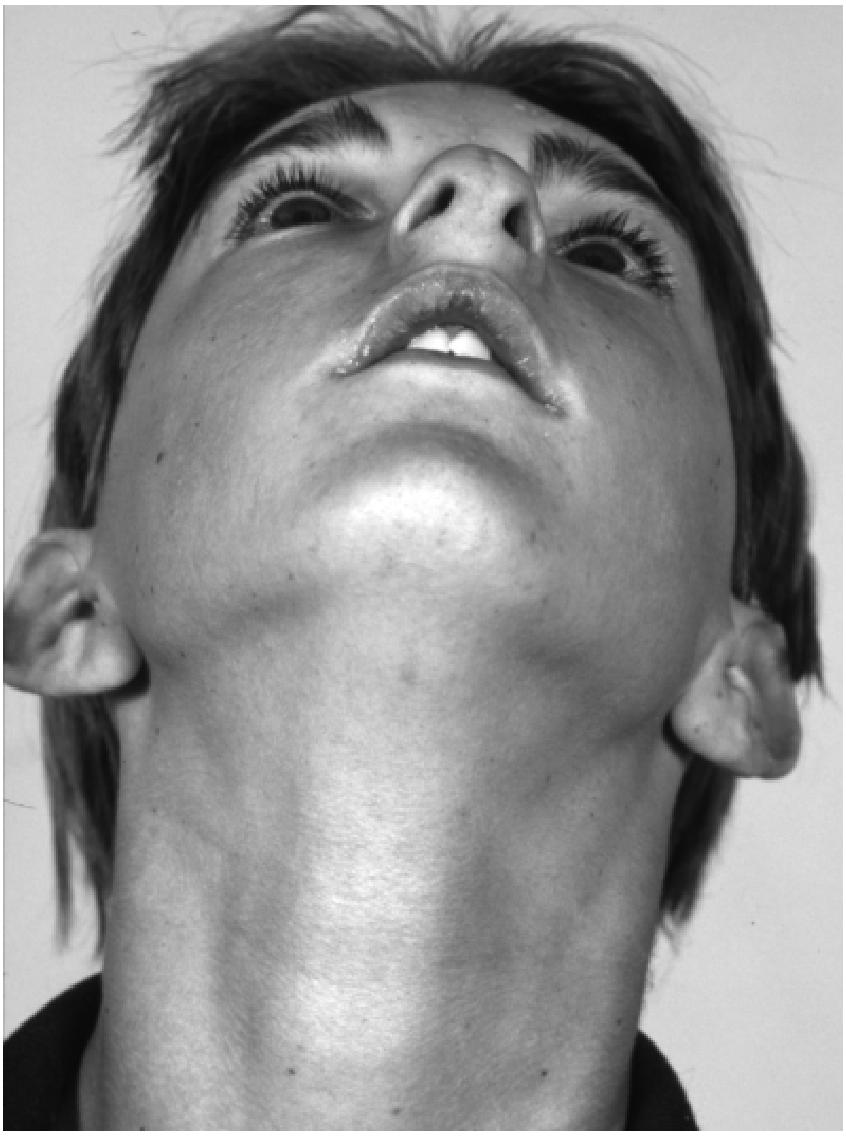
Figure 4



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A proportional reduction in nasal airflow might therefore be expected. Clinically however, reduction in nasal airflow has not been a significant problem even with the maxilla superiorly repositioned eight to ten mm.[45]

Le Fort I superior maxillary movement produces changes in external nasal dimensions most commonly in the alar base width. This change may be undesirable in patients with good pre-surgical nasal dimensions. However, the typical candidate for Le Fort I superior movement frequently has a deficiency in alar width resulting in an improved aesthetic result. This increase in alar width may also be functionally desirable.[45]

The nasal valve area, accounts for most of the inspiratory resistance to airflow. The increase in alar width as a result of this surgery also presumably increases the width of the nasal valve. Thus, the increase in alar width produced by Le Fort I superior movement may open the nasal valve paradoxically reducing nasal airway resistance while the maxilla is repositioned superiorly into the nasal cavity. Guenthner et. al., showed a reduction in nasal airway resistance of 30 per cent in eleven patients treated with the Le Fort I maxillary impaction.[45]

Orthognathic surgery involving a standard Le Fort I osteotomy in combination with a sagittal split ramus osteotomy for advancement of the maxilla and mandible has proven to be very successful in the management of OSAS.[46,47] Success rates in the treatment of OSAS of these studies are between 61 per cent and 100 per cent.[46,47]

Cleft Palate Patients

Although many studies have associated nasal obstruction and hypertrophied adenoids with changes in craniofacial growth, another group of patients who frequently have posterior nasal obstruction are those with cleft palates. These patients often have pharyngoplasty to reduce their nasal airflow during speech.[48]

The pharyngeal flap is performed to improve the quality of speech in the hypernasal cleft palate patient through a reduction in velopharyngeal port size. A secondary effect of this reduction is a simultaneous increase in nasal airway resistance, which may contribute to the dentofacial growth changes seen in children with cleft palates.[48]

Studies that have measured craniofacial growth in cleft patients following a pharyngoplasty were able to demonstrate retarded forward maxillary growth, and an opening mandibular rotation resulting in significant retrognathia. However, both of these studies lack pre-surgical and post-surgical airflow data.[48,49] Conclusions drawn about the cause and effect of upper airway resistance and craniofacial growth therefore relies on the assumption that increased airway resistance occurred in studies following surgery. The case for direct cause and effect is also complicated by the wide range of individual responses in nasal-resistance changes following pharyngoplasty.

Figure 5

Conclusion

Following a review of the literature, it seems evident that determining respiratory pattern is a complex issue. The inability to reliably identify nasorespiratory function and quantify the degree of obstruction has meant there remains no compelling evidence to support a causal relationship between oral respiration and facial growth nor that treatment modalities actually modify the mode of respiration.

Longitudinal studies involving quantitative analysis of breathing pattern at repeated intervals are essential in future research to determine the inter-relationship between upper airway and craniofacial growth.

Figure 6

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coming events

- 11[th] World Congress on Dental Trauma. Oslo, Norway. 21-24 June, 2000. Contact PLUS Convention Norway AS, C. Sundtsgate 10, N-5004 Bergen

- 1[st] World Congress of the International Cleft Lip & Palate Foundation. Zurich, Switzerland. 1-5 July, 2000. Contact Professor Hermann Sailer, University of Zurich, Raemistrasse 100, Zurich CH-8091 Switzerland

- IADR Australian & New Zealand Division. Perth, Western Australia. 2-7 July, 2000. Contact Dr David Booth, School of Oral Health Sciences, 179 Wellington Street, Perth WA 6000

- 88[th] FDI Annual World Dental Congress. Paris, France. 29 November-2 December, 2000. Contact Mr Paul Wilson, FDI World Dental Federation Congress & Exhibition, 7 Carlisle Street, London, England W1V 5RG.

- 30[th] Australian Dental Congress. Brisbane, Australia. 4-8 May, 2001. Contact Congres Secretariat, PO Box 1280 Milton, Qld. 4064 (ada2001@im.com.au)

- 9[th] International Congress on Cleft Palate and Related Craniofacial Anomalies. Göteborg, Sweden, 24-28 June 2001. Contact Conference Secretariat, Congrex Göteborg AB, Box 5078, SE 402 22 GÖTEBORG, Sweden.

- 89[th] FDI Annual World Dental Congress. Kuala Lumpur, Malaysia. 16-19 September, 2001. Contact Mr Paul Wilson, FDI World Dental Federation Congress & Exhibition, 7 Carlisle Street, London, England W1V 5RG.

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